IOWA STATE UNIVERSITY
Digital Repository

Volume 37 | Issue 1 Article 8

1975

Pseudorabies in Swine

Les Meier
Iowa State University

Pseudorabies in Swine

by
Les Meier*

Summary

A producer in north central Iowa experienced a swine epizootic of pseudorabies. The young piglets exhibited typical central nervous signs of trembling, convulsions, and paddling. Mortality was over 50% in the piglets. Pregnant sows showed increased abortions and stillbirths, finally causing a low 5.4 farrowing average. Diagnosis was by virus isolation, fluorescent antibody tests, and histopathology. No treatment was successful. A review of current literature follows with the case reports.

Introduction

Pseudorabies is an acute, infectious viral disease of swine which is of growing importance in the U.S.[4] Formerly thought to be of economic importance only in Europe, it is now causing more significant losses in Iowa and other areas of high swine numbers. Recently further research has led to increased knowledge of its pathogenesis and clinical symptoms, and to the extent of its geographical distribution.[6] Normally only 6 cases of pseudorabies occur in Iowa per year. As of July, 1974, over 34 cases of pseudorabies have been reported. One of the cases is detailed below

Case Report

In north central Iowa, a hog producer was nearing the end of his spring farrowing in June, 1974. The farrowing was in a completely enclosed, controlled environment farrowing building. Nine late sows and their litters were moved out of the building before weaning to allow for cleaning and disinfection. They were placed in a pen in an open front confinement building which also had boars, pregnant sows, and fattening hogs. Through the fence contact was present. Eight days after the move, the 10–21 day old pigs started to show clinical signs of pseudorabies. Out of the 80 pigs from the 9 sows, only 32 survived. The sows never did become very sick. Some of the sows showed vomiting, anorexia, some slight incoordination and nasal discharge. With the next farrowing in August, continued high losses were present. The producer only averaged 5.4 pigs from over 50 sows.

Initial diagnosis was by the area veterinarian when the family's dog died after eating some of the dead pigs. Confirmation was by the Iowa State Diagnostic Laboratory with virus isolation, fluorescent antibody test, and histopathology.

Discussion

The baby pigs which died showed typical signs of pseudorabies for their age. Clinical signs observed in the piglets were depression, anorexia, vomition and diarrhea, trembling, incoordination, spasms of opisthotonus and prostration.[1,3] Some paddling movements were observed and the temperature ranged from normal to 106°. Age has been observed to greatly influence the severity of clinical signs. Generally, pigs under 2 weeks have a high mortality while those over 4 weeks usually show a low mortality.[1] Also the particular strain of virus is important as some strains are more virulent for all ages.[1] Newborn pigs are the most susceptible, with mortalities of 100% being common. These pigs

* Mr. Meier is a fourth year student in the college of Veterinary Medicine at Iowa State University.

usually show no clinical signs.[3] They simply sicken, become prostrate, and die. The commonly seen myoclonia congenita may be related to an infection of pseudorabies.[10] In one herd of 15 sows, all the piglets exhibited the myoclonia congenita symptoms. Two out of three pigs examined were positive for pseudorabies.[11]

In sows and gilts, this infection may be masked under the name of SMEDI. Stillbirth, mummification, embryonic death, and general infertility have all been associated with pseudorabies infections. In a mild case the sows will become anorexic, depressed, constipated, and pyrexic (106°). A mild case can be so transient as to not be noticed clinically. In a more severe infection, pregnant sows may abort, have stillbirths, mummified fetuses, or reabsorb the fetuses depending on the stage of pregnancy. In work done here at Iowa State by Kluge and Maré, 12 gilts were exposed to the virus intranasally.[10] Five died with signs of fever, anorexia, sneezing, coughing, CNS signs, vomition and pruritus at postmortem exam these gilts also displayed fibrinopurulent upper respiratory tract lesions. The other 7 survivors shed the virus for 15 days post exposure. Usually about 50% of infected sows abort. Those less than one month along are most susceptible. The ISU work also dispelled another widespread belief about the disease. Former literature reported that swine never show the pruritus that is characteristic of the disease in other animals. Most of the exposed gilts had a persistent generalized pruritus.[11] Repeated testing for ectoparasites or dermatophytes was negative. This increased pruritus has also been seen in fattening pigs where the disease is normally much less apparent.

Usually pigs of greater than 40 pounds recover without any great loss. But, depending on the strain, the losses can go as high as 35%. The disease can come and go in an entire herd in as little as 11 days and be entirely subclinical.[4] If any clinical signs are seen in these pigs, they are depression, anorexia, pyrexia of up to 106°, head pressing, circling, and some blindness. It should be emphasized that the respiratory changes can entirely overshadow the CNS changes. Certain strains of the virus have been shown to be much more pathogenic to lung tissue than was previously believed.

Pathogenesis

This disease is believed to most commonly enter the pig via the nasal pharynx from nose to nose contact. Once in the nasal pharynx, it rapidly multiplies and remains in the nasal secretions for 10 to 17 days. Approximately 48 hours after infection, the virus reaches the brain via the epineural lymphatics of the olfactory, trigeminal and glossopharyngeal nerves. It then spreads rapidly throughout the brain and spinal cord.[1] It persists in the brain for approximately 10 days, which corresponds with the rise in the body's antibody titer. The respiratory form requires a proper strain of the virus which multiplies rapidly in the lungs to produce a severe and widespread pneumonia.[1]

The most commonly accepted and major mode of transmission between swine is nose to nose contact. Other modes such as rats and airborne transmission have been considered minor. The virus is shed in feces and urine of infected pigs, as well as in the milk of carrier sows. Infected boars at service shed the virus, but the importance of this isn't known for sure. Other hosts such as rats, cattle, cats, dogs, horses, mink, foxes, and raccoons are thought to be dead-end hosts and only capable of serving as mechanical carriers.[1,3]

The main reservoir of the virus is the swine population. Although unproven yet, much evidence points to the existence of carrier pigs capable of shedding the virus following periods of stress.[1,14,8,4] Stresses could be weather, concurrent diseases, or shipping. Sows are especially likely carriers. Even though immune from a previous infection, another strain or the same strain can still reinfect the nasal epithelium for a period of 17 days before being expelled.[1] From these sows then, the piglets can receive the infection when they are most susceptible. The virus has also been shown capable of surviving several months in dried tissue, and for approximately two months in infected premises.[9]

Immunity

A solid immunity to that particular strain follows recovery from infection. The sow can pass this immunity on to the pigs. This passive immunity will last 3–7 weeks. All sows don't develop the same level of antibodies, and a highly contaminated environment may overwhelm the passive immunity level. ". . . neither vaccination nor colostral antibody prevent the excretion of virulent virus: their effect is to prevent illness and not infection . . ."[1] High levels of systemic antibody give little local protection at the nasal epithelium level. Antiserum has been used on baby pigs to reduce death losses. Over all results have been partially disappointing as only a low level of local protection is established in the nasal mucosa.[1]

Vaccines of both the inactivated and modified form have been used. Generally the inactivated types haven't been successful. The live vaccines have induced better immunity and are in general use in Europe. Several different modified strains exist. One of the more common is the K strain.[1] It seems to stimulate local immunity quite well, although the systemic titer stays low. The bad point about the live vaccines is that they insure the virus will be maintained in the population. Also their use is attended by the necessity for all swine to be vaccinated because the presence of only a few non-vaccinated animals results in epizootics.[5] Immunity ". . . doesn't prevent subsequent colonization of the upper respiratory tract by virulent virus, nor the excretion of the virus."[1]

Diagnosis

Diagnosis is on the basis of clinical signs, histopathology, virus isolation, and fluorescent antibody tests of proper tissues. Clinical signs of muscle tremors, incoordination, convulsions with general depression and pyrexia are suggestive in baby pigs. Very young pigs of course may just sicken rapidly and die in a coma without any of the above signs. An increase in the number of stillbirths and abortions are tipoffs in pregnant sows and gilts.

Pathologically, there may be 2–3 mm. foci of necrosis on the liver, spleen, lung, and tonsils. Generally, though, the gross lesions at necropsy are few unless the respiratory form is present. Then the upper respiratory tract can become covered with a thick yellow fibrinopurulent exudate.[10] Laboratory confirmation should reinforce any field diagnosis. Histopathology shows a non-suppurative meningo-encephalomyelitis. A positive fluorescent antibody test of either tonsillar tissue, olfactory bulb, or pons and virus isolation from the brain or nasal swabs of acutely ill older pigs are often successful.

Differential

This disease must be differentiated from transmissible gastroenteritis (TGE), porcine viral polioencephalomyelitis, hog cholera, salmonellosis, Glasser's *E. coli* septicemias, erysipelas, gut edema, and salt poisoning. The gross lesions of most of the above aid in diagnosis. Rabies, although rare in pigs, must be ruled out. Streptococcal meningitis of baby pigs and hypoglycemia also can give nervous symptoms which are confusing. In sows, SMEDI, Parvo virus, leptospirosis, and brucellosis can give similar reproduction problems. Paired serum samples, immunofluorescence and careful observation may aid in diagnosis. Usually virus can not be isolated from mummified pigs or the vaginal discharge.

Prevention and Control

Control of this disease without vaccination requires good management. A closed herd is a good beginning, as carrier pigs are the most common form of introduction. Isolation of new breeding stock before introduction to the herd is essential. Comingling the new stock with the rest of the herd at least 4 weeks before breeding will reduce losses. Separation of the sick from the rest of the herd will prevent new cases, especially in the pregnant and very young. Any dead pigs should be properly disposed of to minimize the chance of spread. Disinfection procedures may prevent transferring the virus around the

farm. For the young pigs, weaning at an early age will greatly reduce the susceptibility, as the sow is the most likely source for them.

Conclusion

Pseudorabies is an old disease now emerging with increased importance in swine production. It will probably become more economically significant before a proper method of control is established.

Bibliography

1. Baskerville, A., Dow C., McFerran, J. B. 1973. Aujezsky's Disease in Pigs. *The Veterinary Bulletin*, 43.9.
2. Blood, D. C., Henderson, J. A. 1971. *Veterinary Medicine*. Third edition. William and Wilkins, Baltimore. Pp. 524–527.
3. Dunne, H. W. 1964. *Diseases of Swine*. Second edition. Iowa State University Press, Ames. Pp. 273–284.
4. Dunne, H. W. Editor 1970. *Diseases of Swine*. Third Edition. Iowa State University Press, Ames. Pp. 337–347.
5. Gustafson, D. P., Kanitz, C. L., Mitchell, M. V. 1972. Immunologic Approaches to the Control of Pseudorabies. *JAVMA* 160:623–633.
6. Hutyra, Marek, Manninger 1938. *Pathology and Therapeutics of the Diseases of Domestic Animals*. Alexander Eger, Chicago. P. 313.
7. Howarth, J. A., DePaoli, A. 1968. Enzootic of Pseudorabies in Swine in California. *AVMA* 152:1114–1118.
8. Howarth, J. A. 1969. A Serologic Study of Pseudorabies in Swine. *JAVMA* 154: 1583–1589.
9. Jubb, K. V. F., Kennedy, P. C. 1970. *Pathology of Domestic Animals Volume 2*. Second edition. Academic Press, New York. Pp. 416–417.
10. Kluge, J. P., Mare, C. J. 1974. Swine pseudorabies: Abortion, Clinical Disease, and Lesions in Pregnant Gilts Infected with Pseudorabies Virus (Aujezsky's Disease). *American Journal of Veterinary Research* 35:911–915.
11. Mare, C. J., Kluge, J. P. 1974. Pseudorabies Virus and Myoclonia Congenita in Pigs. *JAVMA* 164:309–310.
12. Siegmund, O. H., Editor 1973. *The Merck Veterinary Manual*. Fourth edition. Merck & Co., Rathway p. 237.
13. Ziercke, A. V. September 21, 1974. *Personal Communications*.